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CONGENITAL DIAPHRAGMATIC DEFECTS
Salvatore Battiata, M.D., Joseph W. Peabody, Jr., M.D., and Edgar W. Davis, M.D. 29

SKULL DEPRESSIONS IN NEWBORN INFANTS
John Tkacz, M.D. 45

SPEECH AND SPEECH THERAPY FOR A CHILD WITH A CLEFT PALATE
Anthony L. Rotolo, M. A. 50

THE EDITOR'S COLUMN: THE HIGH OBJECTIVE 55

BOOK REVIEW 56

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Congenital Diaphragmatic Defects

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Although the incidence of congenital diaphragmatic hernias has not changed appreciably over the years, occurring about once in 1,200 births (Bowers et al.), the chance for such an infant's survival has been greatly improved. Credit for this gain goes in part to the pediatrician, who, through a greater awareness of the condition and its grave prognosis, is now diagnosing the lesion earlier and advocating surgery more promptly. However, the improved outlook has also paralleled technical advances in pediatric surgery, anesthesiology and fluid and electrolyte management.

Of 127 cases of congenital diaphragmatic hernia collected by Latta in 1922, 88 were stillborn or died within an hour or two of birth. In 1925, Hedblom found an alarming tendency toward conservative management of these infants despite the fact that 75 per cent of babies so handled were dead before the end of the first month of life. The value of surgery was next emphasized by Orr and Neff in 1936. From the literature they were able to collect only 17 cases of congenital diaphragmatic hernia in which operation had been performed in the first year of life, and 9 of these patients survived. In 1940, Ladd and Gross reported their own series of 19 operated cases with 12 healthy survivors, an operative mortality of 37 per cent. That same year, Hartzell presented a series of surgical cases with a 50 per cent survivorship. By 1945, Donovan was able to report 17 operated cases with 13 survivors, representing a mortality rate of only 24 per cent. Recent years have seen continued improvement in surgical results, and after accepting for operation even the most desperate cases Gross (1953) has obtained 76 live patients successfully repaired out of a total operated series of 91.

The importance of early diagnosis is evident, but more important still is the need for early surgical intervention, since the major mortality arises not from those who fail to survive operation but from those who die before surgery is even contemplated. The desire to temporize and postpone operation until the baby is in better physical condition, a policy advocated by Roe and Stephens as recently as 1956, is but an invitation to disaster. Medical treatment is futile, and the threat of sudden death is very real. The only rational approach is surgery as soon as the diagnosis is established and the infant can be prepared for surgery.

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Usually symptoms are present at birth. In some instances, however, the initial symptoms are mild, and the patient is several weeks or months of age before any significant abnormality is suspected and diagnostic studies are instituted. This situation is rather strikingly illustrated by the following 3 patients, all of whom showed a paucity of symptoms in the neonatal period and were operated on at 1½, 2 and 5½ months of age respectively. All 3 patients had congenital foramen of Bochdalek-type defects in the diaphragm and all are alive and well at the time of this report.

CASE REPORTS

Case 1.

S. P., a 6 week old white male infant, was the product of an uneventful delivery after a full term pregnancy which was normal except for slight vaginal bleeding in the second month. He was well until one week prior to admission to Children's Hospital when he seemed to be experiencing some abdominal discomfort, as evidenced by his pulling up his legs and crying. Since he was feeding well, these episodes were interpreted as colic. Treatment consisting of sedative drops was instituted, but there was no appreciable improvement.

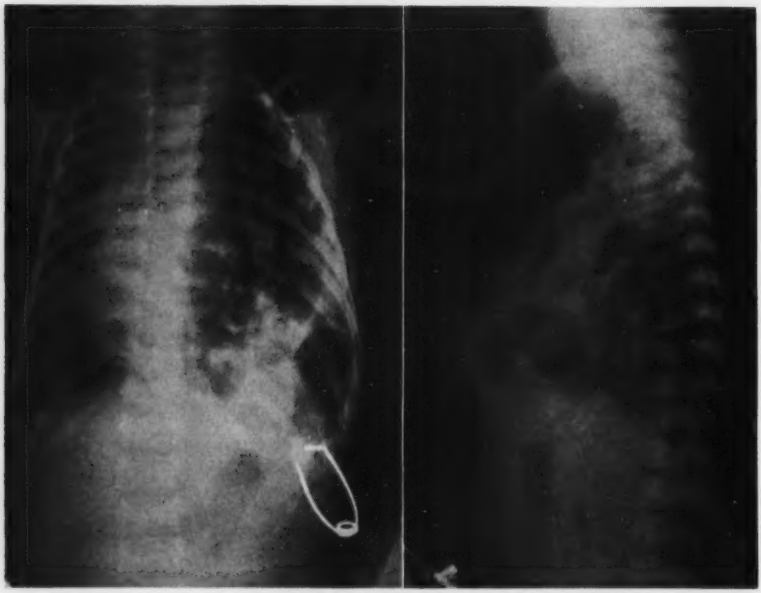

FIG. 1. Case 1: Preoperative chest x-rays showing a bizarre multiloculated cystic element filling the left chest. Note the heart and mediastinum shifted to the right. This was interpreted as a congenital diaphragmatic hernia but the possibility of cystic pulmonary disease could not be excluded.

FIG. 2. Case 1: Barium enema showing splenic flexure, ascending colon and cecum located in the left hemithorax.

During the three days prior to admission the patient vomited twice and his dietary intake progressively decreased; no history could be obtained as to whether feedings were better tolerated in the upright position. The parents described a circumoral cyanosis with crying. There had been no appreciable change in the appearance of the infant's abdomen. His respirations were described as having been rapid since birth.

Admission physical examination revealed a well developed and fairly well nour-

ished infant who was pale and tachypneic and had a minimal circumoral cyanosis. Positive findings included a shift of the trachea to the right; this was accompanied by supraclavicular retraction but by very little intercostal retraction. A resonant percussion note and crackling rales were heard over both lung fields. No bowel sounds were heard in the chest. The heart was shifted to the right as evidenced by the heart sounds being well heard over the right chest.

Chest x-ray in the posterior-anterior and lateral views showed the heart, mediastinum and trachea shifted to the right side (fig. 1). The left diaphragm was not visualized. The entire left lung field was filled with a "circumscribed air-containing area" resembling bowel and suggesting a left-sided diaphragmatic hernia. Since cystic disease of the lung could not be excluded, a barium meal was suggested. The barium passed easily into the stomach and small bowel which were found to be located in their normal positions. Following a barium enema, the splenic flexure, ascending colon and cecum were found to be located in the left hemithorax, displacing the heart and mediastinum to the right (fig. 2). This was felt to be a left-sided diaphragmatic hernia, probably through the foramen of Bochdalek.

At operation the left chest was entered through the seventh intercostal space. Numerous loops of small bowel, as well as transverse and ascending colon with cecum, were found in the chest. There was no sac. Repair was somewhat difficult because of the rather tenuous character of the posterior margin of the defect. A staunch closure was eventually secured by means of figure-of-eight sutures approximating the anterior and posterior rim of the defect. Postoperative course was uneventful except for the occurrence of a pleural effusion which disappeared radiographically by the thirteenth postoperative day.

The patient was readmitted at 5 months of age for a mild gastroenteritis bearing no relation to the previous episode. Chest x-ray at this time was within normal limits.

Case 2.

M. L. S., an 8 week old white male infant, was admitted to Children's Hospital as a feeding problem. He was the product of an uneventful pregnancy. Delivery was described as normal; labor was induced. The birth weight was 6 pounds 13 ounces. The patient was breast fed but did not nurse well. During the first month he vomited frequently, occasionally during feedings as well as several hours later. At times, the vomiting was described as projectile. At one month of age, when he weighed 7 pounds 6 ounces, he was started on a proprietary milk formula. Since there was no improvement in the feeding problem and since, in addition, his stools became loose, he was changed to a high protein formula. The vomiting seemed to stop and the stools became normal. However, the patient continued to tolerate not more than 2 ounces at each feeding. At 8 weeks of age, his weight was still only 8 pounds, and hospitalization was advised.

Admission physical examination revealed a poorly nourished infant who appeared to have no abnormal physical findings. Blood sugar and electrolytes were normal. There was no excess starch or fat in the stool. Bilateral subdural tap did not reveal any subdural fluid. The initial diagnostic impression was pylorospasm.

Chest x-rays revealed the stomach and intestinal tract to be in the left chest (fig. 3). The heart and mediastinum were displaced to the right. This gave the appearance of a left diaphragmatic hernia or an agenesis of the left diaphragm.

At operation through the seventh left intercostal space, the stomach and intestines were found within the left chest and were encased in a thin hernial sac. After reducing the hernia, a 4 cm. defect was found in the posterolateral diaphragm consist-

FIG. 3. Case 2: Figures 3a and 3b are preoperative chest x-rays showing stomach and large bowel filling the left side of the chest. These films were taken five minutes apart during which time the baby burped. Note the marked decrease in size of the gastric gas bubble, emphasizing why these infants may suddenly die from gastric distention. Figure 3c is a portable chest x-ray taken on the first postoperative day.

ent with a foramen of Bochdalek defect. The sac was incised, and the anterior and posterior margins of the defect were brought together by many figure-of-eight and mattress sutures of heavy black silk. In so doing, another rent in the diaphragm appeared posteriorly where the diaphragm was extremely thin and friable. This second diaphragmatic opening was easily repaired, after which the sac was split open and plicated in two separate layers so as to buttress the primary closure of the diaphragmatic defect. The left lung was slightly hypoplastic and filled only one half of the left pleural space. The postoperative course was uneventful.

Case 3.

A. H., a 5 month old white female infant, had been previously operated upon at Children's Hospital when 2 months of age for an ileocecal intussusception. On that occasion there had been nothing in the examination or at operation to suggest a diaphragmatic hernia. No chest x-ray had been obtained.

Since that admission the mother noted that the baby had grunting respirations and seemed short of breath upon lying down, especially after feedings. There was no nausea or vomiting. Chest x-ray showed partial opacification of the right pleural space. The liver could be delineated above the diaphragm, and a barium swallow showed small intestine and colon also to lie in the right chest (fig. 4). When told of these findings the mother then realized that she could feel peristaltic waves in the chest.

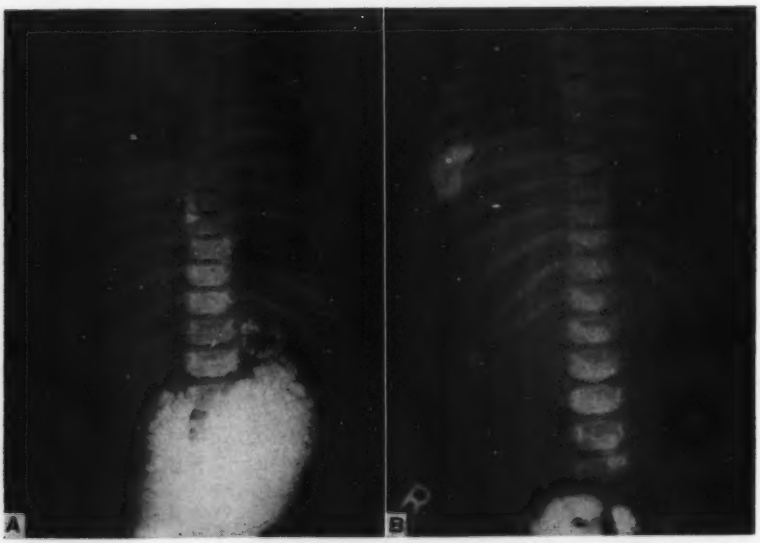

FIG. 4. Case 3: Fig. 4a is a chest x-ray obtained several hours after barium swallow and reveals barium-filled loops of small intestine in the right hemithorax. Fig. 4b is an x-ray taken 24 hours later at which time a segment of colon is outlined above the right leaf of the diaphragm. The solid mass in the right lower hemithorax represents liver.

Physical examination showed the left lung to be clear. Breath sounds were diminished over the right chest. No note was made as to the presence or absence of bowel sounds over the chest.

At thoracotomy via the right eighth intercostal space, many loops of small intestine, the liver, colon and part of the stomach were found in the right chest. There was no sac. A relatively large posterolateral Bochdalek-type defect was delineated; there was a very little rim along the lateral margin which extended to the chest wall. After the hernia was reduced, closure was effected by approximating the anterior and posterior margins with heavy interrupted silk sutures. The lateral-most portion of the defect could not be satisfactorily obliterated through this approach, but by entering the chest at a second site two interspaces lower, the lateral side of the defect could be securely sutured to the chest wall. The lung expanded completely. Recovery was uneventful.

DISCUSSION

Pathogenesis and Classification of Congenital Diaphragmatic Defects

The diaphragm is a single, independent, dome-shaped muscle arising from the circumference of the lower part of the thorax, which, when normally formed, completely separates the abdominal and thoracic cavities.

In order to understand better the pathogenesis of this condition, a brief review of the embryology of the diaphragm will be helpful.

There are four embryonic components which fuse to form the normal diaphragm. The septum transversum, or central portion of the diaphragm, originally arises opposite the third and fourth cervical vertebrae, but migrates caudally, and at about the third week of fetal life, is located opposite the twelfth thoracic vertebra. An outgrowth of the dorsal mesentery then fuses with the septum transversum and forms the median dorsal portion. Two openings at the posterolateral aspects of the developing diaphragm are known as the pleuroperitoneal canals. These are normally closed at about the fourth fetal week by a double layered serous membrane consisting of pleura and peritoneum. This pleuroperitoneal fold, developed from the dorsal mesentery and the Wolffian ridge, forms what is known as the pleuroperitoneal portion of the diaphragm. The lateral component of the diaphragm arises from the lateral body wall and ultimately attaches to the periphery of the pleuroperitoneal fold. During the third fetal month, muscle mass followed by the phrenic nerve invades the membranous diaphragm, the posterior part and crus of which are transformed into muscular structures (fig. 5).

Whether or not the diaphragmatic hernia is covered by a sac depends upon the time the hernia forms. If developing bowel moves into the chest and prevents the complete formation of the diaphragmatic leaf, the coelomic cavity remains undivided. This occurs in 90 per cent of cases. If the bowel migrates cephalad after the membranous diaphragm forms, but before muscular invasion occurs, there will be a hernial sac with a partial

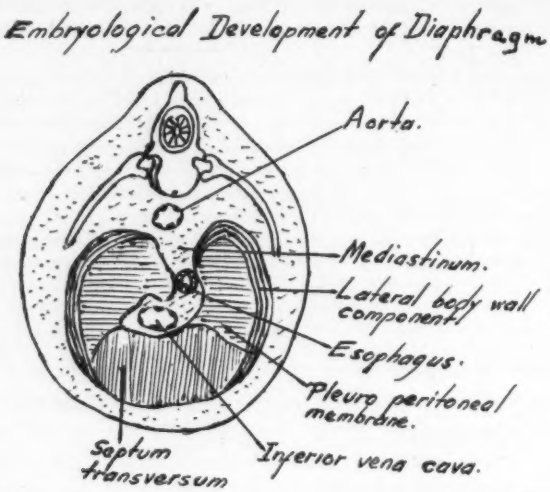

FIG. 5. Embryological development of the diaphragm. After Broman

muscular covering. If the muscular development is imperfect or incomplete, eventration may occur. On the other hand, there may be an isolated defect which under stress will later result in a hernia. Those hernias with a sac are referred to as "true hernias," and those without a sac "false hernias."

There are several classifications of diaphragmatic defects based on both anatomical and developmental considerations. The classification of Gruber is based on the development of the diaphragm, i.e., agenesis of the diaphragm, failure of union of the diaphragmatic anlagen, failure of muscular development, and rarely, displacement of the viscera resulting from dystopia of the diaphragm secondary to agenesis of the lung.

Ladd and Gross classify the diaphragmatic defects as follows:

1. Hernia through the foramen of Bochdalek
2. Esophageal hiatus hernia
   a. Short esophagus and thoracic stomach
   b. Normal esophagus and thoracic stomach with diaphragmatic defect
3. Hernia through the vena caval or aortic openings
4. Agenesis of part of or the entire leaf of the diaphragm
5. Inflammatory necrosis of the diaphragm
6. Hernia secondary to trauma
7. Hernia through the foramen of Morgagni
8. Eventration of the diaphragm

The muscular structure of the diaphragm in the adult is divided into three parts according to their origin: sternal (the weakest), costal, and lumbar (the strongest). All insert into the margin of a central tendon.

Hernias of the foramen of Bochdalek are due to defects in the posterolateral portion of the diaphragm, and result from persistent pleuroperitoneal canals. This is the most common site of herniation (fig. 6). Ninety per cent of these hernias do not have a sac. The left side of the diaphragm is five times more commonly involved than the right, partly because the presence of the liver on the right impedes herniation.

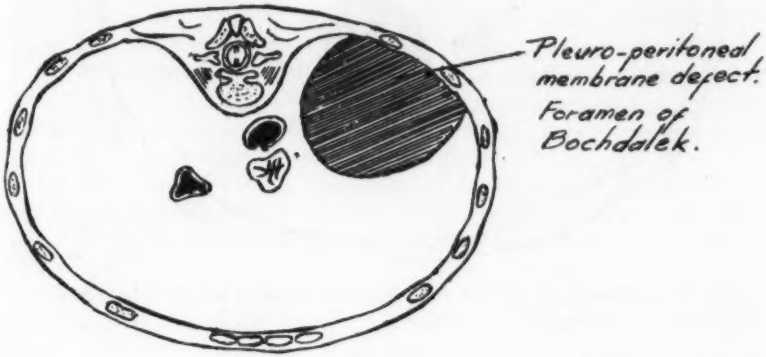

FIG. 6. Diaphragmatic defect at the pleuro-peritoneal canal (foramen of Bochdalek). From Arey: Developmental Anatomy, 5th edition, 1954, Philadelphia, W. B. Saunders Company.

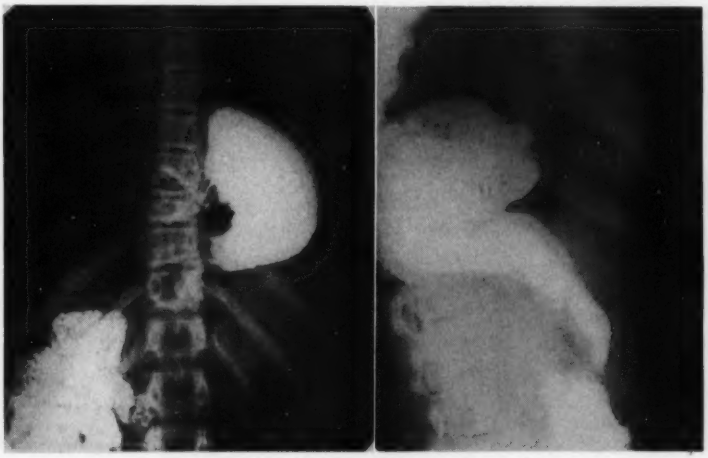

FIG. 7. Hernia through esophageal hiatus. Barium swallow of a 15 year old white girl with upper gastrointestinal symptoms since infancy. The entire stomach lies above the diaphragm.

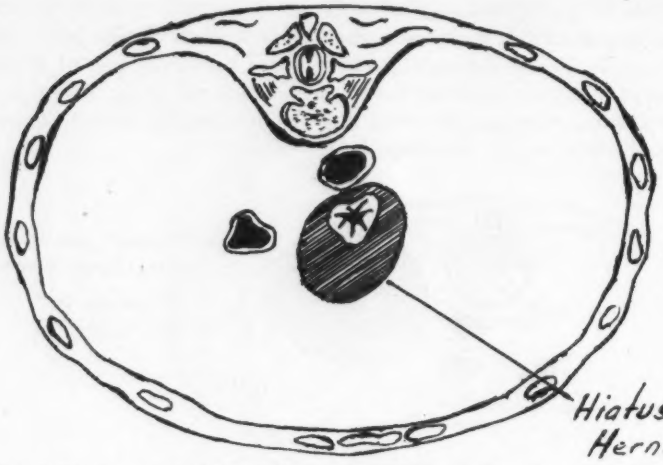

FIG. 8. Diaphragmatic defect at the esophageal hiatus. After Arey: Developmental Anatomy, 5th edition, 1954, Philadelphia, W. B. Saunders Company.

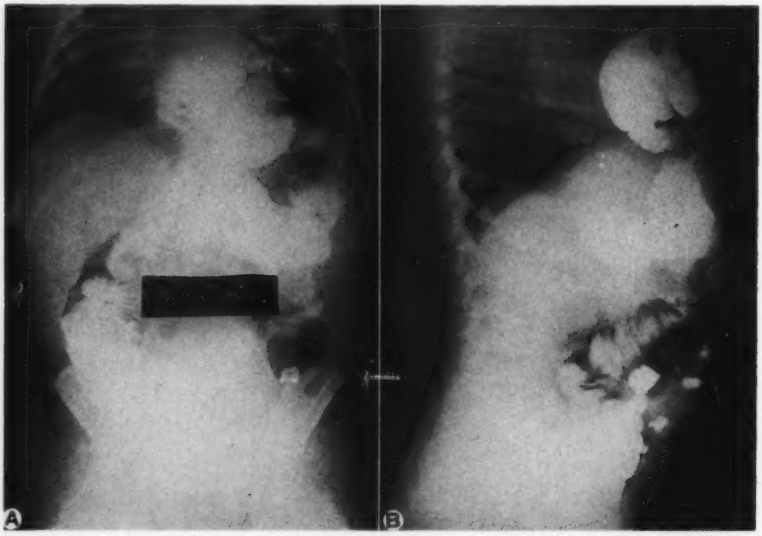

FIG. 9. Hernia through the foramen of Morgagni. Barium enema in a child with herniation of the colon into the chest via a retrosternal defect.

Both types of esophageal hiatus hernia usually have a sac which limits the upward progression of the abdominal viscera. The sac seldom contains more than stomach, and usually does not produce symptoms in children (figs. 7 and 8).

Agenesis of the diaphragm in its entirety, or a failure of muscular development with herniation at or before birth, is rare. According to some authorities, absence of the diaphragm is incompatible with life and does not lend itself well to surgical correction. Correction can be attempted with cutis graft or tantalum mesh.

Inflammatory necrosis with perforation of the diaphragm and herniation may be secondary to a subdiaphragmatic abscess or erosion of the diaphragm by a thoracotomy tube left in place for too long a period of time, for example, during drainage of chronic empyema.

Traumatic rupture of the diaphragm or evisceration of the abdominal contents into the chest may be secondary to a crushing injury to the abdomen. The site of rupture may well be at a congenitally weak point in the diaphragm.

On each side of the xiphoid attachment of the diaphragm are two spaces filled with areolar tissue and covered with pleura and peritoneum. The

FIG. 10. Diaphragmatic defect at the retrosternal area. After Arey: Developmental Anatomy, 5th edition, 1954, Philadelphia, W. B. Saunders Company.

internal mammary arteries pass through these openings which are known as the foramina of Morgagni or Larrey's spaces (figs. 9 and 10). Because of the small potential space between the sternum and the attachment of the pericardium, these hernias are never extensive.

Eventration of the diaphragm is described as an abnormally elevated position of one leaf of an intact diaphragm, as a result of paralysis, aplasia, or atrophy of the muscle fibers. The etiology of eventration is obscure; it is present at birth although it may produce no symptoms. It is frequently noted as an incidental finding. Eventration may be due to injury to the phrenic nerve at birth. Damage to the phrenic nerve will result in paradoxical motion, as compared to normal motion (as slight as it might be) of an eventrated diaphragm with an intact nerve supply. Eventration of the diaphragm secondary to aplasia of the muscle fibers may be difficult to distinguish grossly from agenesis of the muscular fibers with a peritoneal-pleural sac covering the abdominal viscera. Diagnosis is made by x-ray and fluoroscopy. Some authorities feel that eventration of the diaphragm should not be operated on if the patient is asymptomatic and the defect discovered only on routine examination. There is no medical treatment; if symptoms are present, surgery is indicated.

Symptomatology

The symptoms arising as a result of a diaphragmatic hernia are produced by the presence of abdominal contents in the thoracic cage. This causes a decreased negative intrathoracic pressure on the involved side and compression of the lung which results in poor pulmonary function on the affected side. The reduction in pulmonary function may be great enough to produce cyanosis on even mild exertion. Acute symptoms may develop from an intestinal obstruction secondary to a volvulus.

The severity of symptoms will depend upon the amount of abdominal contents in the thoracic cage, whether there is kinking or constriction of a hollow viscus and whether the lung is collapsed.

A common complaint is cyanosis which may be evident immediately after delivery and persist, or may be transient and appear only during nursing or crying. At birth the need for prolonged resuscitation in the presence of a good heart beat, with subsequent feeble respirations and dyspnea, may be indicative of a diaphragmatic hernia.

Vomiting following feeding is occasionally encountered and may be decreased by holding the patient in the upright position, provided there is no mechanical obstruction. The esophageal hernias seldom produce symptoms in infancy, except where there is a free reflux of gastric juice causing an esophagitis, with reflex esophageal spasm and dysphagia. Melena and hematemesis may be sequelae of such an esophagitis. Constriction by the

diaphragm of a partial thoracic stomach may cause obstruction and bleeding. Hernias through the foramen of Morgagni seldom produce symptoms; those that are produced are due to partial compression of a hollow viscus.

Physical findings

There is invariably an increase in the cardiac and respiratory rate. Percussion of the affected hemithorax will give a dull or tympanitic note, depending on what the underlying structures are and whether gas is trapped in the bowel. The mediastinum, trachea, and heart are pushed to the opposite side. Because of the greater incidence of left-sided diaphragmatic hernias, dextrocardia has been frequently described. There may be absent or distant breath sounds on the affected side; bowel sounds may also be heard. Respiratory movements on the affected side are usually diminished. There is usually dehydration and marked loss of subcutaneous tissue.

Diagnosis

It is difficult to make a diagnosis of diaphragmatic hernia from the history alone. A history of cyanosis, dyspnea and vomiting should suggest a diaphragmatic hernia. The only physical finding which can positively confirm the diagnosis is the presence of bowel sounds in the chest. If these are not heard, the diagnosis must be made by x-ray. In any event, a chest x-ray should confirm the diagnosis. Roentgenographic findings include the presence in the thorax of haustral markings and small collections of gas; sometimes a nonhollow viscus such as the liver is observed. In addition, one may see a decrease in the gas pattern in the abdomen and absence of the diaphragm. The aforementioned shift of the mediastinum, trachea and heart to the opposite side may also be noted.

In confirming the diagnosis, it is important that contrast media be avoided as much as possible. If their use is absolutely necessary, a thin barium solution should be used since thick barium may make reduction of the hernia difficult at surgery. If barium is used, it should be aspirated after the x-ray study is completed.

The main problem in differential diagnosis is confusion radiographically with congenital lung cyst, hypertrophic lobar emphysema and pneumatoceles. Dextrocardia may also be simulated. Moore and co-workers have pointed out that the characteristic diagnostic triad in all infants diagnosed in the first few hours of life is one of dyspnea, cyanosis and "dextrocardia."

From the standpoint of symptomatology congenital diaphragmatic hernias can be confused with any of the many causes of breathing problems in the newborn, including brain injury and agenesis, subdural hematoma,

hyaline membrane disease, laryngeal anomalies with respiratory obstruction, massive atelectasis, spontaneous pneumothorax, tracheoesophageal fistula, mediastinal cyst, etc.

It cannot be stressed too strongly that any child suffering from persistent or repeated attacks of dyspnea, cyanosis, vomiting, cough and gastrointestinal upset should have an adequate examination to rule out the possibility of a diaphragmatic hernia.

Treatment

There are divergent opinions as to the best surgical approach for the repair of these defects. Gross favors an abdominal approach, as do many others. Like Koop and Johnson, we prefer a thoracic incision. Exposure is eminently satisfactory. Reduction has been no problem in our hands, and the presence of a sac has been useful in providing a buttress line in the repair. From below, the presence of a sac is often difficult to ascertain inasmuch as it is closely applied to the lung. If the defect is closed without obliteration of the sac a cyst will be created above the diaphragm.

On the other hand, the abdominal approach does permit simultaneous correction of any coexisting anomaly beneath the diaphragm. Also, when the abdominal viscera do not fit back into the peritoneal cavity due to their long residence in the chest, it is possible to settle for a ventral hernia and simply close the skin over the viscera. The ventral hernia can then be repaired at a later date. So far we have had no difficulty in reducing all of the viscera into the abdomen. Moreover, the exposure from above seems much more satisfactory and permits better reconstitution of the diaphragm which is often extremely thin and easily torn (case 2). We suspect that in this particular case a complete and permanent repair would have been impossible from the abdominal side.

Although crushing the phrenic nerve has been advocated by some as a means of relieving strain on the sutured diaphragm and also permitting easier reduction of the abdominal contents, we feel very strongly that this is an extremely risky maneuver and should be avoided. These infants need an intact diaphragm to assist with ventilation and permit the raising of bronchial secretions. A paralyzed hemidiaphragm strikes us as a dangerous complication and is quite unnecessary.

Surgical correction should be attempted as soon after the diagnosis is made as is practical. Dehydration and electrolyte imbalance must be corrected, if necessary, before surgery. Anemia may be corrected with small transfusions. Preoperative and postoperative gastric suction should be maintained to prevent gastric dilatation, vomiting, and possible aspiration. Adynamic ileus may delay oral feedings, and if prolonged, may be treated with neostigmine.

In the past six years, there have been 14 cases of diaphragmatic defects diagnosed at Children's Hospital. Ages at diagnosis varied from 36 hours to 4½ years. There were 4 cases younger than 1 month and 8 cases less than 6 months of age. The diagnosis was made by x-ray in all but 4 cases. In 5 of the 14, the diaphragmatic defect was an incidental finding, the x-ray having been taken for suspicion of a foreign body or respiratory infection. Two of the cases presented are in the latter category.

Only 12 of these children had surgery. One patient with traumatic diaphragmatic hernia died before surgery could be performed; another child with eventration of the diaphragm did not go to surgery.

Of the cases operated on, 4 were due to diaphragmatic eventration; 2 died (mortality rate 50 per cent). There were 4 cases of hernia through the foramen of Bochdalek with one death (mortality rate 25 per cent). Of the total of 12 cases undergoing surgery, there were 3 deaths, an over-all mortality rate of 25 per cent. Postoperative complications encountered were intestinal obstruction, pneumothorax, and pulmonary edema. The operative approach was transthoracic in all cases.

SUMMARY

Congenital diaphragmatic defects may cause symptoms days or weeks after birth, and present with other than the classical symptoms one usually associates with this disorder.

Three unusual cases of diaphragmatic hernia are presented.

It is of the utmost importance that once the diagnosis is made, immediate corrective measures be undertaken.

The records of the Children's Hospital of the D. C are reviewed, and the experience with congenital diaphragmatic defects for the past six years is presented. In over 30 per cent, the diagnosis of congenital diaphragmatic defects was an incidental finding when x-rays were taken to rule out other conditions.

We would like to thank Hugh C. Clark, M.D., Preston A. McLendon, M.D., and Frank J. Murphy, M.D. for allowing us to present the above cases.

BIBLIOGRAPHY

ARNHEIM, E. E.: Congenital hernia of diaphragm with special reference to right-sided hernia of liver and intestines, Surg., Gynec. & Obst. **95:** 293, 1952.

BAUMGARTNER, C. J. AND SCOTT, R. F.: Surgical emergency of diaphragmatic hernia in infancy, Arch. Surg. **61:** 170, 1950.

BELSEY, R. AND APLEY, J.: Congenital right sided diaphragmatic hernia; some difficulties in differential diagnosis and operative repair, Arch. Dis. Childhood **24:** 129, 1949.

BISGARD, J. D.: Congenital eventration of diaphragm, J. Thoracic Surg. **16:** 484, 1947.

Bowers, V. M., Jr., McElin, T. W., and Dorsey, J. M.: Diaphragmatic hernia in newborn; diagnostic responsibility of obstetrician, Obst. & Gynec. **6:** 262, 1955.

Boyd, D. P.: The transthoracic repair of esophageal hiatus hernia, S. Clin. N. America **36:** 631, 1956.

Campbell, S. M.: Symposium on thoracic surgery; anaesthesia in thoracic surgery, S. Clin. N. America **34:** 1007, 1954.

Chisholm, T. C., Spencer, B. J., and Wyatt, O. S.: Symposium on accidents and emergencies; intestinal obstruction in early infancy, Pediat. Clin. N. America **1:** 907, 1954.

Clinton-Thomas, C. L.: Congenital diaphragmatic hernia in premature infant, Lancet **1:** 1155, 1955.

DeNicola, R. R. and Vracin, D. J.: Diaphragmatic hernia through foramen of Morgagni, J. Pediat. **36:** 100, 1950.

Donovan, E. J.: Congenital diaphragmatic hernia, Ann. Surg. **122:** 569, 1945.

Geever, E. D. and Merendino, K. A.: Repair of diaphragmatic defects with cutis grafts; experimental study, Surg., Gynec. & Obst. **95:** 308, 1952.

Greenwald, H. M., and Steiner, M.: Diaphragmatic hernia in infancy and in childhood, A. M. A. Am. J. Dis. Child. **38:** 361, 1929.

Gross, R. E.: Congenital hernia of diaphragm, A. M. A. Am. J. Dis. Child. **71:** 579, 1946.

———: The Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1953.

——— and Ferguson, C. C.: Surgery in premature babies; observations from 159 cases, Surg., Gynec. & Obst. **95:** 631, 1952.

Hartzell, J. B.: Diaphragmatic hernia in children; resume of 68 cases occurring in children under 10 years of age treated by operation, Am. J. Surg. **48:** 582, 1940.

Harrington, S. W.: Symposium on abdominal surgery, S. Clin. N. America **30:** 961, 1950.

———: Diagnosis and treatment of various types of diaphragmatic hernia, Am. J. Surg. **50:** 381, 1940.

Joynt, G. H. C., and Laird, R. C.: Symposium on thoracic surgery; thoracic trauma, S. Clin. N. America **34:** 951, 1954.

Kirklin, B. R.: The roentgenologic diagnosis of diaphragmatic hernia, Collect. Papers Mayo Clin. **39:** 622, 1947.

Koop, C. E. and Johnson, J.: Transthoracic repair of diaphragmatic hernia in infants, Ann. Surg. **136:** 1007, 1952.

Ladd, W. E. and Gross, R. E.: Congenital diaphragmatic hernia, New England J. Med. **223:** 917, 1940.

Latta, J. S.: Congenital deficiency of the diaphragm, Am. J. Dis. Child. **24:** 297, 1922.

Mayo, C. W. and Remington, J. H.: Acute surgical conditions of the abdomen; congenital lesions, Collect. Papers Mayo Clin. **38:** 145, 1946.

Moore, T. C., Battersby, J. S., Roggenkamp, M. W. and Campbell, J. A.: Congenital posterolateral diaphragmatic hernia in the newborn, Surg., Gynec. and Obst. **104:** 675, 1957.

Orr, T. G. and Neff, F. C.: Diaphragmatic hernia in infants under one year of age treated by operation. J. Thoracic Surg. **5:** 434, 1936.

Rickham, P. P.: Strangulated diaphragmatic hernia in neonatal period, Thorax **10:** 104, 1955.

Roe, B. B. and Stephens, H. B.: Congenital diaphragmatic hernia and hypoplastic lung; a clinical evaluation, J. Thoracic Surg. **32:** 279, 1956.

SANFORD, M. C.: Congenital diaphragmatic hernia, Clin. Proc. Child. Hosp. **8:** 252, 1952.
SWYER, P. R.: Progress in pediatrics; partial thoracic stomach and esophageal hiatus hernia in infancy and childhood, A.M.A. Am. J. Dis. Child. **90:** 421, 1955.
THOMSON, S. A.: Symposium on thoracic surgery; diaphragmatic hernia in infancy and childhood, S. Clin. N. America **34:** 997, 1954.
TRUESDALE, P. E.: Diaphragmatic hernia; report of cases illustrating its varieties, South. Surg. **8:** 1, 1939.
WYATT, O. S.: Congenital diaphragmatic hernia, Journal-Lancet **61:** 164, 1941.
ZELLER, W. E.: Diaphragmatic hernia in infancy, West. J. Surg. **58:** 619, 1950.

Skull Depressions in Newborn Infants

JOHN TKACZ, M.D.*

Skull depressions may occur in the newborn as a result of pressure on the head during the birth process. These are sometimes called "depressed fractures of the skull" although there is usually no actual fracture. The asymptomatic skull depression in the newborn presents the problem of whether or not surgical elevation should be done.

The increased elasticity of the calvarium of the infant, the bones of which are separated only by the membranous sutures, permits alteration in the shape of the head during delivery without the danger of skull fracture. In addition, at birth the bones of the cranial vault as a rule contain less calcium than shortly thereafter. The calcium content of the skull is directly proportional to the size of the fetus; therefore, bending of the cranial bones and green-stick fractures are more common. On the other hand, comminution is rare. The indented bone may spring back immediately or may gradually resume its normal contour. Blood vessels traversing the inner aspect of the skull are not embedded in the bone at this age and thus escape injury.

Fractures of the cranial vault in children are very frequent while fractures of the base are rare. The explanation lies in the resiliency of the sutures lines and the relatively lighter construction of the base of the skull; thus fractures of the vault do not tend to radiate to the base. In addition, a less rigid spine in children does not favor the transmission of trauma to the base of the skull when vertebral injury has occurred.

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Two important questions arise in the consideration of any skull fracture: 1) whether it is compound and apt to infect the brain and its membranes, and 2) whether it is so depressed that the indented bone produces pressure on the brain. The importance of the skull injury, therefore, is its effect on the underlying brain. Conversely, however, it is possible that head trauma may cause no demonstrable skull injury and yet cause extensive damage to the soft structures within the skull.

The soft, nonresilient substance of the brain makes it very vulnerable to any trauma applied to the skull. White matter is more susceptible to injury than gray matter, and therefore damage is often more extensive in the central portion of the brain. The pathology of brain injury is similar to that produced by trauma to other soft tissues of the body and includes evidence of edema, hemorrhage, and necrosis. The degree of these changes depends on the nature of the particular injury. In addition to the effect of local trauma, the entire brain is subjected to pressure due to swelling at the injury site and the limited intracranial space. Compensatory adjustments such as a decrease in the normal amount of cerebrospinal fluid, blood, and tissue fluid within the skull may prevent severe brain damage. However, this process of compensation has limits, and when these are exceeded, intracranial pressure increases and the vital centers in the medulla may become damaged or destroyed by the resulting ischemia.[1]

The newborn's "depressed fracture" (skull depression) is not a true fracture, since the actual continuity of the bone is not broken. Instead, this "fracture" consists of a snapping in of a thin resilient portion of bone.[2] These injuries are comparable to green-stick fractures of the long bones in childhood[3] and can result from pressure against the maternal symphysis pubis,[2, 4] pressure against the promontory of the sacrum,[2, 4, 5] forceps pressure,[3, 6-8] digital pressure by the obstetrician at the time of delivery,[3] or deformed pelvis;[8] they occur particularly during breech deliveries.[7]

These skull depressions look as if the bone had been pressed in by the outer aspect of the bowl of a spoon, and vary from a centimeter in diameter to the marked depression of an entire frontal or parietal bone (fig. 1). The larger depressions are usually more troughlike than spoon-shaped. Bilateral parietal depressed fractures may occur as a result of difficult forceps extraction.[3] Skull depressions generally occur in the parietal[5, 7, 9] or frontoparietal[10] region.

The subject of skull depressions has been treated in the literature by a variety of specialists: the pediatrician, obstetrician, neurologist, neurosurgeon, pathologist, surgeon, and orthopedist. This may account for the many contradictions present, an example of which is evident in the reporting of incidence. The incidence ranges, according to the author read, from "commonly seen,"[3] "may occur,"[2, 4, 8] "sometimes occur,"[6] "not common,"[11] to "rare."[9]

There is even confusion as to what the abnormality should be termed. This is apparently due to attempts to use terms which do not suggest the presence of actual fracture. Various authors have described these lesions as "indentation without fracture,"[12] "derby hat or rubber ball skull depressions,"[6] "depressed fractures without a break in continuity,"[2-4] "groove skull depression,"[7] "depressed fracture,"[8, 9, 13] and "spoon-shaped or troughlike depressions."[10]

An actual fracture can occur with a skull depression. The presence of symptoms with skull depression would dictate surgical intervention whether or not visible fractures were present. It might be expected that skull depressions are accompanied by bleeding on the inner aspect of the skull or that direct damage to the brain with the eventual formation of scar tissue would result, but it seems, in fact, that any injurious result is most excep-

FIG. 1. Roentgenographic examination of the skull in the antero-posterior position showing a skull depression in the left parietal bone.

tional.[3, 6, 9] Of course the absence of a depression or fracture of the skull in an infant does not rule out brain injury; as Watson-Jones[5] points out, even severe intracranial birth injuries accompanied by asphyxia, respiratory distress, or circulatory failure, and cerebral hemorrhage with spastic diplegia are usually unaccompanied by skull fracture.

In the newborn infant with the asymptomatic skull depression, what will be the fate of the depressed area of bone? More important, what will be the fate of the underlying cortex? Will spontaneous elevation of the bone occur or will surgical elevation be necessary? If surgical elevation is indicated, when should it be done? These constitute the main problems in determining the course of management.

Very occasionally uncorrected depressions persist into adult life;[6] no treatment is necessary unless it is thought desirable to improve the cosmetic appearance by raising a bone flap. Some authors believe that long-continued pressure might cause localized external compression and injury to the underlying cortex.[2-4] This may serve not only to interrupt function, but may also interfere with growth and perhaps establish an epileptogenic focus.

Controversy in the literature revolves around whether these depressions should be surgically elevated[3] or whether they should be allowed to elevate spontaneously.[5-7, 10, 11] One reason for the difference of opinion may be that the former group is composed largely of neurosurgeons who are being consulted only for the more marked depressions. If it is thought that a cosmetic defect will persist, this in itself would be an indication for elevation of the depression, as it could conceivably prevent unnecessary psychological trauma when the child grows older.

Surgical correction is variously recommended from about the first day or two of life[3] to the second or third month of life.[9] Newborn infants tolerate this type of surgery very well. Prematurity is no contraindication to the operation, as long as the baby's temperature is controlled.[3] Various recommended procedures include elevating the depression by pulling or prying upward with a hook or a small periosteal elevator.[8] Rogers[12] states that the best way to deal with the indented skull of the infant is to cut out a flap of bone with a pair of scissors, reflect it at its base as a hinge, press up the depressed area with the thumbs, and replace the flap. Ingraham and Matson[3] drill a small burr hole in normal bone and introduce a narrow blunt instrument such as a periosteal elevator under the depressed bone which is then gradually levered back into position. Occasionally it "pops" back in one quick motion, but usually it is necessary to maneuver it back more slowly by changing the position of the elevating instrument several times. If the depressed bone is impacted so tightly that it will not move with reasonable pressure, or if it can be elevated but not held in normal position,

they believe it is wise to make a linear cut in the bone with stout scissors from the burr hole to the center of the area of depression. This will allow the fragments to be elevated without difficulty.

Williams[14] considers the location and the amount of depression in determining his treatment. If the frontal area is involved, he repairs the skull. If the depression is in the parietal area and slight, no repair is done, as in his experience spontaneous elevation will occur; if the depression is more than slight, surgical elevation is done. His reasons for elevation are to prevent brain damage and to assure a good cosmetic result; he considers these reasons equally important. He prefers to do corrective surgery as soon as the baby can tolerate it well, and feels that the second or third day of life is not too soon if the baby is well stabilized.

Williams does not use a burr hole, as the resulting skull defect is, in his opinion, unnecessarily large. Instead he "scratches" a small linear defect with a sharp instrument into the skull (this is easily done because of the skull's softness), and then inserts the elevating instrument through the defect.

SUMMARY

Skull depressions, also called by some physicians "depressed fractures of the skull" although there is usually no actual fracture, may occur in the newborn as a result of pressure on the head during the birth process.

Asymptomatic skull depressions in the newborn infant raise the question of whether or not surgical elevation should be done. Spontaneous elevation of the depression may occur. If elevation does not occur, the cortex underlying the depression may be subjected to damage from constant pressure, or the cosmetic defect of a skull depression may persist.

There is much difference in opinion among physicians of the various specialties concerning the incidence of skull depressions in the newborn, the indications for their treatment, and the time and method of treatment. The neurosurgeons, judged by the data reviewed, are seemingly least prone to wait and hope for spontaneous elevation. With the passage of time, the danger of brain injury increases and the difficulty in elevating the depression becomes more difficult.

REFERENCES

1. Montgomery, A. H.: Head Injuries, in Brennemann's Practice of Pediatrics, Hagerstown, Maryland, W. F. Prior Company, Inc., 1948, vol. 4, chap. 22.
2. Potter, E. L.: Pathology of the Fetus and the Newborn, Chicago, Year Book Publishers, 1952.
3. Ingraham, F. D., and Matson, D. D.: Neurosurgery of Infancy and Childhood, Springfield, Illinois, Charles C Thomas, 1954.
4. Rawl, A. E.: Depressed skull fracture in utero; a case report and review of the literature, J. S. Carolina M. Ass. **53**: 44, 1957.

5. Watson-Jones, R.: Fractures and Joint Injuries, ed. 4, Baltimore, The Williams and Wilkins Company, 1952, vol. 1.
6. Blount, W. P.: Fractures in Children, Baltimore, The Williams and Wilkins Company, 1955.
7. DeLee, J. B.: The Principles and Practice of Obstetrics, ed. 7, Philadelphia, The W. B. Saunders Company, 1940.
8. Nelson, W. E.: Textbook of Pediatrics, ed. 6, Philadelphia, The W. B. Saunders Company, 1954.
9. Norman, A. P.: Birth trauma, Brit. M. J. **1**: 37, 1956.
10. Grulee, C. G., and Eley, R. C.: The Child in Health and Disease, a textbook for students and practitioners of medicine, ed. 2, Baltimore, The Williams and Wilkins Company, 1952.
11. Jolly, H.: Birth injuries, Practitioner **176**: 369, 1956.
12. Rogers, L.: Simple depressed fracture of the skull, Brit. M. J. **1**: 1431, 1953.
13. Holt, L. E., and McIntosh, R.: Holt Pediatrics, ed. 12, New York, Appleton-Century-Crofts, Inc., 1953.
14. Williams, Jonathan: Personal communication to the author.

Speech and Speech Therapy for a Child with a Cleft Palate

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If we analyze the activity of speech closely we realize that it is not a basic physiological activity of the human being, but rather is an overlaid or trained function which is made possible by the individual's ability to modify and alter his behavior and thereby produce a system of communication which assumes meaning for him. This is an imitative skill acquired by the individual and is almost totally dependent upon the vegetative functions of chewing, sucking, swallowing and respiration.

Speech is not a skill achieved at the age of 18, 24 or 36 months but one which is developed from the very first evidence of respiration and continued on through a long series of developmental stages or levels. These stages of speech development include such activities as babbling, vocal play, jargon, partial words and finally enunciation of whole words and phrases. There is much that actually precedes the words for which we wait. The progression from the early, primarily reflexive sound utterances of the infant to the

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complex process commonly referred to as adequate verbal communication or adult speech normally requires a span of seven or eight years of early life to be fully realized. Therefore, it becomes clear that this is a very intricate process.

The acquisition of so called "normal" speech is dependent upon a number of factors, principal among which are such items as the presence of an audible system of communication for the individual to imitate, adequate intellectual ability, normal auditory acuity, good auditory discrimination, good auditory memory span, an intact and well functioning central nervous system, a well balanced emotional being, sufficient neuromuscular control, coordination of the anatomical structures and functions required for speech and an intact and well functioning physical organism.

A deviation in any one or more of these areas will make itself known in the quality or quantity of a child's speech performance. It is for this reason that, regardless of how minor the cosmetic impediment of a cleft lip and/or palate, it is seldom, if ever, a hidden anomaly. Its presence is revealed by the individual as he engages in sound production and makes attempts at verbal communication.

Although a cleft palate is initially classified primarily as a cosmetic and feeding problem, it most accurately falls in the category of a speech abnormality. The greatest degree of speech difficulty experienced by the child will be in the areas of resonance and articulation of the individual speech sounds. Since a cleft of the palate, regardless of the specific type and/or degree, leaves incomplete the bony and muscular separation of the oral and nasal cavities, the proper direction of the emission of air during the formation of individual and combined speech sounds becomes a serious problem for the child with such a defect. Without adequate closure between the nasal and pharyngeal resonating chambers a problem of nasalized speech will result.

It is to be pointed out here that the nasal cavity is very infrequently used as a resonating chamber for speech in the noncleft palate individual. In fact, there are only three sounds in the English phonetic system that require the nasal cavity to serve any function at all; these are the sounds, *m*, *n*, and *ng*. In all other sound production nasal resonance is prevented by closure of the nasopharyngeal port.

In addition to the problem of hypernasality excessive articulation errors are a frequent source of difficulty in the speech of children with a cleft palate. Since articulation takes place in the oral cavity, it is easily understood how abnormalities of the tongue, jaw, palate and lips, plus irregularities of dentition and occlusion will make themselves known in the articulation.

Unfortunately, the mere surgical, orthodontic or prosthodontic correction

of the palatal condition does not entirely eliminate the problem of speech difficulties in most cases. There may be other residual organic or functional abnormalities of the pharyngeal musculature, or deficiency of palatal tissue. It is also important to note that all speech abnormalities observed in the child with cleft palate are not of necessity related to the palatal condition itself. It is estimated that 5 per cent of the over-all (noncleft palate) general school age population have speech disorders severe enough to interfere with social or emotional adjustment or educational progress. Problems of reduced auditory acuity, poor general physical or emotional well being, and the child's general attitude toward speech and general level of speech performance must be considered, as well as his reaction to the act of speaking. It is commonly seen that the child with a cleft lip and/or palate displays a mixture of speech defects due variously to 1) delayed speech and language development, 2) malformed dental structures, 3) hearing loss, and 4) the condition of the palate or lip itself.

It is estimated that approximately 80 per cent of the children with a cleft palate are retarded in articulation skills to some detectable degree and that over 50 per cent show a retardation of two years or more. There is considerable inconsistency in the articulation of the consonant sounds by these children. However, they are generally articulated incorrectly 60 per cent of the time. The greatest degree of difficulty is experienced with the fricative and voiceless consonants and the least with the nasal consonants. These children appear to be somewhat retarded in the mean length of their verbal responses and scope of vocabulary usage but are not retarded in the structural complexity of their remarks.

Aside from problems in the area of speech, significant additional anomalies are said to occur in approximately 10 to 25 per cent of the cleft palate population. There is a tendency for these children to be of less than average intelligence although the degree of retardation is not considered to be serious. The literature reports a greater incidence of otitis media among these children. Statistically the percentage of these children with concomitant hearing problems is listed by some researchers to be as low as 25 per cent and by others to be as high as 90 per cent. Obviously, a hearing problem, when present, is a factor of considerable importance in these children.

A child, such as the child with a cleft lip and/or palate, who lacks the necessary physical apparatus for normal speech development is handicapped *from the moment he tries to produce isolated sounds* and not at the age of 18 to 36 months when the use of whole words and phrases are initiated. Many professional persons interested in the repair or correction of the cleft condition feel little need to consider surgery, prosthetics, speech therapy, etc. before the age of 3 years. The reason for this is given to be that

a child does not begin to master the consonant sounds until this age. However, it must be pointed out that the child of 3 years has engaged in two and a half or more years of experimentation in the production of both vowel and consonant sounds which then allow for the mastery of the consonant sounds from the age of 3 years on. Consequently, if adverse conditions are allowed to exist prior to the age of 3 years they will make themselves felt by limiting the amount of generalized speech skill which the child can develop prior to this age, and delay the development of subsequent speech development. This, along with other medical factors, should be an important consideration in the timing of surgery, orthodontia, prosthodontia, speech therapy, etc. for this group of children.

The question most frequently asked the speech therapist by other professionals regarding management of the cleft palate child is the age at which the child should be referred to the speech therapist. It is questionable how much, if any, *specific* speech therapy should be attempted with the infant or child under the age of 2½ or 3 years. However, this does not mean that the therapist has no service to offer the cleft palate child and his parents prior to this time. Realizing that the resulting speech difficulties will be the most handicapping aspect of the cleft palate child's problem, and also of most concern to his parents, it would seem advisable to include the speech therapist on the "habilitation team" soon after the parents have been informed of the condition. Often through mere general counseling sessions the therapist is able to inform the parents what to expect from the child in the way of speech performance, to ease their anxieties regarding the child's delayed speech development, and to provide from time to time specific activities which can be attempted in the home to assist the child toward maximum speech proficiency in light of the palatal condition. The mere reassurance provided the parents of the child by being informed at the start that there are professionally trained persons within the community who will be of assistance to him would seem reason enough to include the therapist much in advance of the actual therapy sessions being considered.

In providing the child with a cleft lip and/or cleft palate with a program of speech therapy, the over-all goals are to assist him in developing the structures and functions involved in the speech process to the level of their maximum potential and to teach compensatory as well as natural speech manipulations and activities that provide the child with as serviceable a system of communication as possible. The specific activities or techniques used in the therapy program with a child with a cleft palate are a very individual matter and are dependent not only on the nature and degree of the cleft but also upon the individual who possesses the cleft, his

responsiveness, personal makeup, and upon any concomitant problems which might also be present.

In order to be of the most possible assistance to the child the therapist must first make an intensive and extensive evaluation of the individual's level of general speech intelligibility. Once this has been determined, the speech errors observed must be judged as to the degree of their relationship to the cleft condition and whether they are maturational or functional in character. Those speech errors which are considered to be directly related to the palatal condition must then be analyzed as to the demands they make on the general speech musculature.

During the course of the therapeutic process itself the therapist will direct the majority of his efforts along three specific lines. The first goal would be to make the child with a palatal condition aware of and responsive to sound in general. It is for this reason that the therapist would hope to see the child and parent early in the rehabilitative process and not initially when the child has reached the age of 3 or 4 years. There is much general speech development assistance which can be given to the parent and child if contact with the therapist is established at an early enough age. This is not to be misinterpreted as the enrollment of every cleft palate child in an active speech therapy program as soon as the condition has been identified. Next, the therapist would hope to encourage the child to use his speech musculature for all of the nonspeech activities such as sucking, chewing, swallowing, etc. These activities and functions must be encouraged and developed in order to assist in the later development of the child's specific speech skills. A great portion of the therapy program will be assigned to developing discrimination between the various speech sounds and producing these individual sounds and sound combinations as effectively as possible. The final goal, of course, is to extend the child's vocabulary and to give him a means toward, and a sense of success in, the art of communication.

BIBLIOGRAPHY

HAHN, E.: Speech therapy for the preschool cleft palate child, J. Speech & Hearing Disord. **23:** 605, 1958.

KESSLER, H. E.: The Relationship of dentistry to speech, J. Am. Dent. Ass. **48:** 44, 1954.

LILLYWHITE, H.: Teamwork in the Oregon cleft palate program, J. Speech & Hearing Disord. **21:** 18, 1956.

LIS, E. F., PRUZANSKY, S., KOEPP-BAKER, H., AND KOBES, H. R.: Cleft lip and cleft palate perspectives in management, Pediat. Clin. N. America **3:** 995, (Nov.) 1956.

MACCOLLUM, D. W., RICHARDSON, S. O., AND SWANSON, L. T.: Habilitation of the cleft palate patient, New England J. Med. **254:** 299, 1956.

McWILLIAMS, B. J.: Articulation problems of a group of cleft palate adults, J. Speech & Hearing Research **1:** 68, 1958.

PALMER, J. M.: The pharyngeal flap operation: role of the speech therapist, J. Speech & Hearing Disord. **23:** 601, 1958.

RUESS, A. L.: The clinical psychologist in the habilitation of the cleft palate patient, J. Speech & Hearing Disord. **23:** 561, 1958.

SPRIESTERSBACH, D. C.: Criteria for establishing the need for a speech appliance, J. Speech & Hearing Disord. **21:** 365, 1956.

———, DARLEY, F. L., AND ROUSE, V.: Articulation of a group of children with cleft lips and palates, J. Speech & Hearing Disord. **21:** 436, 1956.

———, DARLEY, F. L., AND MORRIS, H. L.: Language skills in children with cleft palates, J. Speech & Hearing Research **1:** 279, 1958.

The Editor's Column

THE HIGH OBJECTIVE

Discoveries in the field of medical science are coming to light at such a pace that hardly a day passes without the newspapers taking note of these accomplishments. The inference drawn from such a state of affairs is that the landmarks on the horizon, once so familiar, are being remolded or replaced, entailing a new orientation on the part of the physician. An incredible revolution has transpired during the last century. It was just one hundred years ago that Virchow, the dedicated disciple of pathologic anatomy, shared his important microscopic discoveries with his inquiring colleagues. With extraordinary care he recorded his findings and attempted to explain organic disease processes through reasoning and his powers of observation. Our debt to him is a continuing one. During his lifetime the world of pathology was bounded by the current knowledge of morbid anatomy, and clinical pathology, as we know it today, did not exist. The ultimate tool of the pathologist then was the high power objective of his microscope. Astute as these clinicians were, this instrument was the court of last appeal, the arbiter for dissenting Aesculapians.

How different is the picture today. Pathology is no longer confined within rigid limits. The artificial barriers that separated pathology have melted in the solvents of pathological research and investigation. The results of these activities are reflected in the rapid strides made in medical diagnosis and treatment. Today pathology belongs to everyone in medicine.

The ultimate tool, the high power objective of the pioneer pathologist, while retaining an important place, can no longer bring into focus all of the details necessary in arriving at a correct diagnosis. The present day pathologist arrives at a diagnosis by compounding the ingredients of information coming to him from many sources. Some of the sources are allied fields such as biochemistry, genetics and biology. If, indeed, "What is past is prologue," the pathologist knows that in time the boundaries which appear to separate related disciplines will also vanish. This would complete the picture—the quest for understanding the complete cycle of natural processes would then be fulfilled. Toward this fulfillment the pathologist makes his contribution. This, then, becomes his new high objective.

G. H. G.

Book Review

Textbook of Pediatrics. Edited by WALDO E. NELSON, M.D. with the collaboration of 81 contributors, ed. 7, 1462 pp., with 428 figs. Philadelphia: W. B. Saunders Company, 1959, $16.50.

As always, Nelson and his contributing authors have turned out an excellent standard reference book in pediatrics. However, any text which attempts to cover an entire field of practice inevitably emphasizes certain areas to the neglect of others, and this book is no exception. The question of emphasis is a personal matter, but it seems to me that in this era of "The New Pediatrics"* the sections on Emotional Development and Psychologic Disorders leave something to be desired. Instead of a dynamically oriented approach which might outline fundamental principles of therapy for the practicing pediatrician, the emotional disturbances are presented in a rather old fashioned "descriptive" manner.

A glowing exception to this approach is the section on Enuresis which gives a nicely balanced presentation of emotional and physical factors and their management. In this chapter is a paragraph which is essential in the practice of any field of clinical medicine. (The italics are the author's):

The diagnosis of an emotional disturbance in a child is not made by

* The New Pediatrics, editorial, Clin. Proc., Child. Hosp. **16**: 1, 1960.

first excluding physical disease; the two conditions are not mutually exclusive. Difficulties in the emotional area are diagnosed by history, observation of the child's behavior and an evaluation of the parent-child relationship.

Among other sections which treat their subjects in a similarly well rounded fashion are those on Convalescent Care, Conservation-of-hearing programs (essentially a well presented editorial) and The Physician and the Child with a Handicap, which discusses the family, institutions, and resources in the community as well as the child's emotional and physical status.

A splendid reminder for the resident and the practicing physician and a detailed outline for the student is the section on Diagnostic Study of Neurologic Disease, which describes a complete technique of history taking and physical examination of children, of infants, of hysterical patients and of unconscious patients. This section is an introduction to a lucidly and logically presented chapter on The Nervous System which is followed by a discussion of Mental Deficiency, Cerebral Palsy, and Behavior Problems Associated with Organic Brain Damage.

Although the average pediatrician has little if any use in his routine practice for the reviews of respiratory physiology, liver function and cardiac catheterization data, these sections offer a good background for the understanding of clinical disease and are well worth reading.

As genetic counseling becomes more important, the physician will find that the section on Prenatal Disturbances provides facts and concepts which can help him give appropriate advice to his patients. Other ready references which make this book an essential in the library of every physician who takes care of children are the section on poisoning, the tables in the appendix, and a table on Selection of Diagnostic Microbiologic Specimens.

GEORGE J. COHEN, M.D.

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